6
OUTPUT STAGE

OVERVIEW

ACE is organised into multiple CS input channels and a common output stage, upon which all of the CS input channels converge. The Neural Multiprocess Memory Model (NMMM) and the CS Trace Circuit (CSTC), which were previously developed separately in Chapters 4 and 5 respectively, may be combined to produce an almost complete CS input channel. This Chapter describes how the individual expectations of reinforcement from each CS input channel are combined to produce the compound "CS expects US" output of ACE, and the way in which the US input is processed in order to determine the compound asymptotic strength of association able to be supported by a US. These are all CS-nonspecific mechanisms, and collectively form the common output stage of ACE. The two signals generated by this output stage are fed back to each input channel to drive the SIM learning rules described in Chapter 7.

EXPECTATION OF REINFORCEMENT

Each individual CS input channel is primarily responsible for acquiring the predictive relationship between its *CS* input, and subsequent delivery (or nondelivery when otherwise expected) of reinforcement. More specifically, each input channel captures the classical conditioning contingency between *CS* and subsequent US presentation, in addition to the temporal relationship between the CS and the US.

If the US input is more likely to occur shortly following CS input activation than at other times, then a positive contingency exists, and the CS input channel produces an excitatory output signal (EXC) in response to CS presentation. Conversely, a negative contingency leads to the production of an inhibitory output signal (INH) from the CS input channel. As illustrated in Figure 6-1, the "CS expects US" output (OUT) is obtained. by subtracting the sum of all inhibitory CS channel output signals (INH) from the sum of all excitatory CS channel output signals (EXC), and then only passing non-negative values.

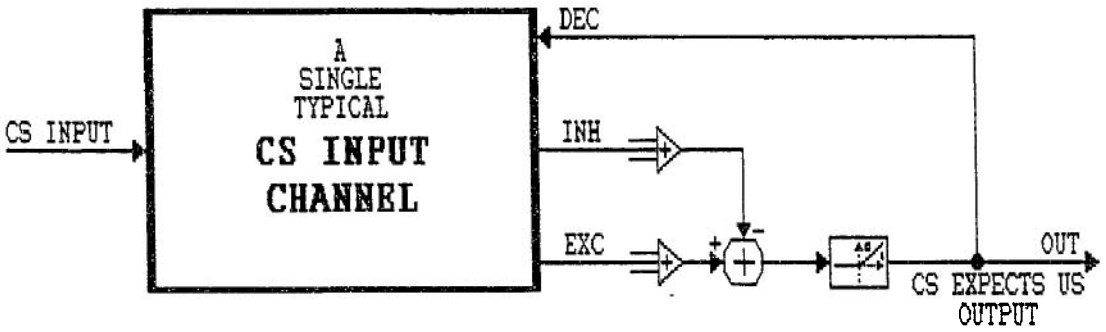

FIGURE 6-1. That past of the output stage which determines the compound expectation of reinforcement, and the way in which it interfaces with a single typical CS input channel. The operators immediately right of both INH and EXC represent convergent summation from each of the *CS* input channels to the single output stage.

In many respects OUT corresponds well with term V in Equation [2-1] of the Rescorla-Wagner model (Rescorla and Wagner, 1972), which represents the compound associative strength of all presented CSs. In ACE, OUT also doubles as the decremental feedback signal DEC. Rejection of negative values is an improvement suggested by Rescorla (1979) that prevents further decreases in associative strength when compound inhibitors are presented, which otherwise occurs in the Rescorla-Wagner model.

Note, however, that ACE is a real-time model dealing with intra-trial processes and interactions, while the Rescorla-Wagner model is a trial-level model that aims to account for the result of each trial only. Hence, OUT represents a phasic, timed expression of the expectation of reinforcement that is capable of driving a correctly timed CR, whereas V is a continuous expression of the strength of association between a *CS* and a US.

Difference Equations 16-11 and [6-2] mathematically define how the OUT and DEC signals are determined from the individual excitatory and inhibitory output signals from each CS input channel.

$$OUT[T] = pos(sum\{N=1 \text{ to } n: EXC[T, N]\} - sum\{N=1 \text{ to } n: INH[T, N]\}) \quad [6\text{-}1]$$

$$DEC[T] = OUT[T] \quad [6\text{-}2]$$

Where: pos(x) = x, if x >= 0.
pos(x) = 0, if x < 0.
N = Number of CS input channels.
T = time state number.
The interval between suscessive time states is 10ms.

It transpires that this part of the output stage is the most conventional mechanism deployed within ACE. In terms of the generation of the output signal, it corresponds to what has for some time been the basic output stage configuration for ANN elements. However, the range of behavior that it is to help support when functioning as an integral part sf' ACE considerably extends its conceptual role. This will become apparent in Chapters 7 and 8.

REINFORCING EFFECT OF THE US

Each CS input channel also responds to actual (as opposed to expected) US input activation via the incremental feedback signal (INC), which is distributed to each CS input channel from the output stage. As is the case with DEC, INC has its intratrial counterpart in the Rescorla-Wagner model.

The reinforcing signal INC corresponds to an intratrial phasic version of the compound asymptotic strength of association supported by the US, which is represented by term L in Equation [2-1]. Each CS input channel may therefore be regarded as the site at which compound associative strength (V) is subtracted from compound asymptotic strength of association supported by the CS (L), in order to determine how individual associative strengths need to be altered to reduce the difference between L and V. The short term retention capability of associative STM within each CS input channel (Chapter 4) enables this comparison to occur, despite the asynchronous nature of CS and US presentation, and the different response shapes of the CSTC output signal (CST, Chapter 5) and the reinforcement feedback signal INC.

Incorporating the essence of the Rescorla-Wagner model within ACE automatically means that stimulus amplitude effects, conditioned inhibition, extinction, overshadowing, compound conditioning and discriminative stimulus effects are able to be supported (in addition to the behavior produced by the NMMM and the CSTC).

US Duration Effects

Experiments studying consummatory classical conditioning of the rabbits' NMR have shown that increasing US duration provides more effective reinforcement, but that progressive increases in US duration provide progressively less additional reinforcement (Ashton, Bitgood, and Moore, 1969; Tait, Kehoe, and Gormezano, 1981).

If the US input were to correspond directly to the INC feedback signal which reinforces CS-US associations in the STM of each CS input channel, then the

above qualitative relationship between US duration and extent of reinforcement would already be essentially supported by the temporal modulation of the reinforcing effect of INC upon STM by RGM. A detailed explanation of the RGM's role and behavior is provided in Chapter 7. Figure 6–3 illustrates, among other things, a typical temporal profile of RGM values that would dominate modulation of acquisition at an ISI of 630ms. The important point to note is that after peaking shortly following US onset (as is consistent with the results of Smith, 1968), RGM gradually declines in amplitude. Thus the integrated effect of INC over time is increased at a diminishing sate by progressive increases in US duration (for US durations in excess of 70ms), producing the desired qualitative behavior. However, quantitatively, this mechanism alone appears to be insufficient.

Ashton, Bitgood, and Moore (1969) studied the effects of US duration and intensity upon delayed conditioning of the rabbits' NMR using a 630ms ISI, and US durations of 50ms and 350ms. Their results show that the extent of acquisition on day 2 to a 350ms US was 1.25, 1.63, and 1.94 times greater than that to a 50ms US (for US intensities of 4, 2, and 1mA respectively). While the ratio diminished to approximately 11 later in conditioning, this may have been due to a ceiling effect. These results therefore indicate that a 350ms US appeared to provide somewhere between 25% and 100% more reinforcement than a 50ms US.

It is clear from Figure 6-3 that if the US input corresponded to INC, which is in turn modulated by RGM, then a 350ms US would be almost 7 times more effective than a 50ms US, as determined by the ratio of the areas under the CST curve over the intervals 630-680ms, and 630-980ms.

In order to provide a more pronounced attentuation of the INC signal within the first 100ms of the US duration, and so provide a better fit with empirical results, a new cumulative quantity will now be introduced which is referred to hereafter as Reinforcement STM (RSTM). As shown in Figure 6–2, RSTM accumulates at a rate proportional to the extent by which the US input amplitude exceeds the RSTM amplitude. This same signal forms the reinforcing feedback signal INC. Thus, as RSTM accumulates, INC diminishes.

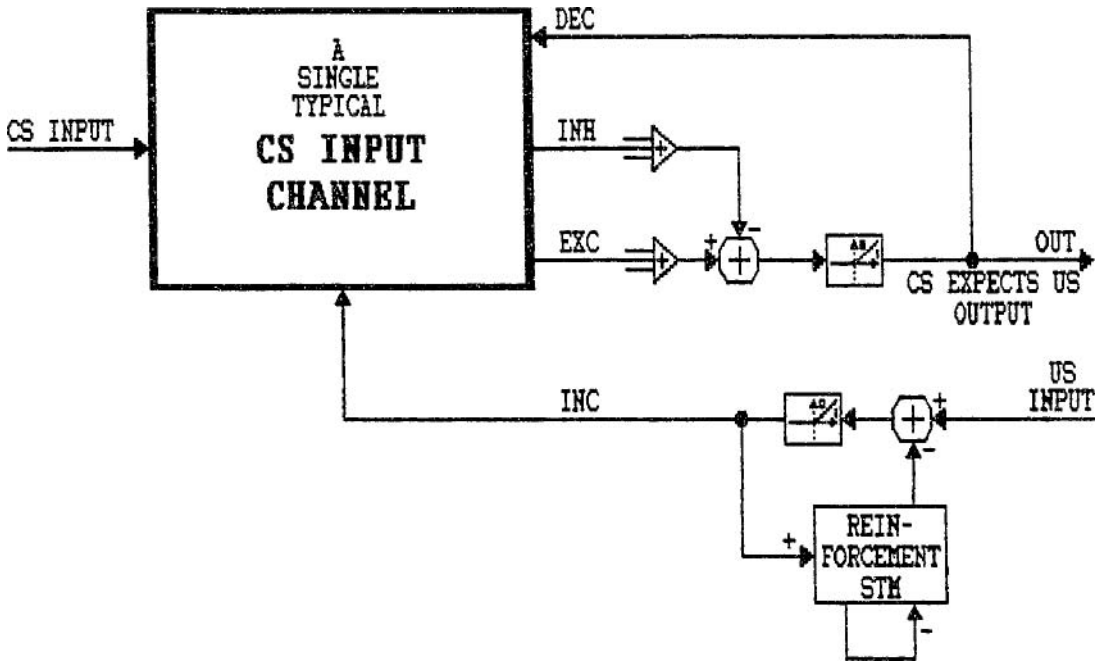

FIGURE 6-2. The complete output stags of ACE, shown interfacing with a single typical CS input channel. The output stage now incorporates Reinforcement STM to appropriately modulate the effect of increasing US duration upon the compound strength of association able to be acquired by the CS input channels.

The difference Equations defining how RSTM and INC are generated are as follows:

$$INC[T] = pos(US[T] - RSTM[T]) \quad [6\text{-}3]$$

$$RSTM[T+1] = RSTM[T] + RSTMacc.INC[T] - RSTMdep.RSTM[T] \quad [6\text{-}4]$$

Where: pos(x) = x, if $x >= 0$.

pos(x) = 0, if $x < 0$.

RSTMacc = accumulation rate of RSTM.

RSTMdep = depletion rate of RSTM.

T = current time state.

T+1 = next time state.

The interval between successive time states is 10ms.

Figure 6–3 illustrates the INC signals and RSTM levels produced by 50ms and 350ms US durations. It is apparent from Figure 6–3 that the area under the 350ms **INC** curve is approximately one and a half times greater than that under the 50ms **INC** curve. This ratio is marginally reduced when the effect of RGM modulation of **PNC** is taken into account. This result, with RSIM used as described above, accords far more favourably with the results of Ashton, Bitgood, and Moore (1969) than when **RSTM** is omitted.

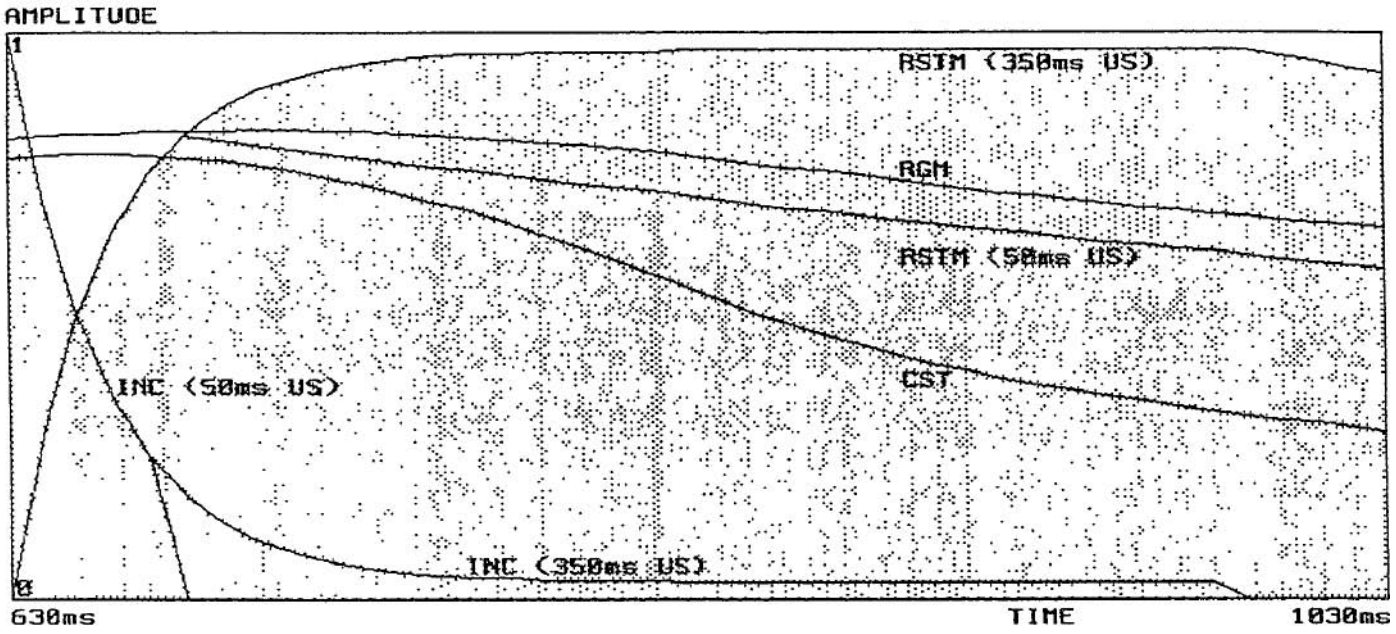

FIGURE 6–3. Reinforcement Short Term Memory **(RSTM)** and reinforcing feedback signal **(INC) 'levels following** US inputs of 50ms and **350ms** duration, commencing **630ms** after CS onset. Also **shown** are **the** CS Trace (CST) and **Reinforcement Gating** Short Term Memory **(RGM)** levels that **peak shortly** after US **onset,** which **are** produced by a 50ms **duration** CS. Equations **16–31 and** [6–43 were used to generate **the RSTM and INC curves, with RSTMacc = 0.30 and RSTMdep = 0.01.**

The simple passive decay **of** RSTM (illustrated below RSIM in Figure 6–2, and defined by the last term in Equation [6–4]) ensures that INC asymptotes towards a positive nonzero **minimum** value, so that increases in US duration in excess of 350ms continue to contribute to the reinforcing effect of the US. This is consistent with the empirical results of Tait, Kehoe, and Gormezano

(1981), in which the percentage of CRs observed continued to increase significantly when the US duration was increased from 1500ms to 6000ms.

The asymptotic minimum level attained by INC when driven by a sustained unity amplitude US is determined by RSTMdep/(RSTMacc+RSTMdep). In order to produce the substantial reduction in INC described above, this asymptotic minimum needs to be very small (much less than 1). This means that RSTMdep << RSTMacc. RSTMacc is primarily responsible for determining the rate of decrease of INC. RSTMdep is then set to provide the appropriate asymptotic level.

US Intensity Effects

Increasing US intensity has the unsurprising effect of increasing both the rate and the strength of conditioning in many classical conditioning experiments studying different types of response systems and species (Mackintosh, 1974, pp. 70–71). More specifically, empirical results obtained using the NMR preparation are also entirely consistent with these US intensity effects (Gormezano, Kehoe, and Marshall, 1983).

It is apparent from Figure 6–2 that the amplitude of the INC feedback signal will be proportional to the amplitude of the US input. Since INC determines the maximum compound strength of association able to be supported by the US, the desired qualitative relationship between CR amplitude and US intensity will be supported. While this relationship is usually more logarithmic than linear (e.g., Smith, 1968), it is assumed that this nonlinearity results from the operation of sensory transducers and sensory preprocessing, and so is not included within ACE.

SUMMARY

The common output stage of ACE supports all interaction between the multiple CS input channels, and is responsible for producing the compound "CS expects US" output of ACE. It also accepts the US input, and after some processing, distributes the reinforcing effect of the US to each of the CS input channels.

Although ACE is a real-time intra-trial neuronal model of classical conditioning, the output stage combines the expectation of reinforcement acquired by each CS input channel, and the actual reinforcement subsequently delivered, in a manner consistent with an improved version of the Rescorla-Wagner model. As a consequence, the considerable behavioral capabilities of the Rescorla-Wagner model are also exhibited by ACE, in addition to the behavior produced by the new NMMM and CSTC systems developed in previous Chapters.

The output stage also includes a specially developed Reinforcement Short Term Memory (WSTM). Computer simulation results are presented which indicate how RSTM is utilised to produce a quantitative relationship between US duration, and its reinforcing effect, that is consistent with empirical results from animal experiments using the NMR preparation. The desired qualitative relationship between US intensity, and its reinforcing effect, is also directly implemented.